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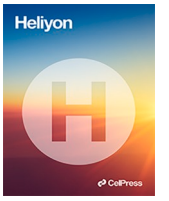

Research article

Clinical and vascular lesion characteristics of the patients with takayasu arteritis manifested firstly as acute myocardial infarction at onset

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ABSTRACT

Objective: To explore clinical and vascular lesion characteristics of the patients with Takayasu arteritis (TA) manifested firstly as acute myocardial infarction (AMI) at onset and to improve the diagnostic rate of TA.
Methods: The clinical and angiographic data of six patients with TA manifested firstly as AMI at onset were retrospectively analyzed.
Results: Of six patients (16–25 years old), 83.33% (five cases) was female, three patients had a history of hypertension and three patients did not have any medical history. One patient had intermittent effort chest tightness. On admission patients all presented with chest pain, dyspnea, hypotension, cardiogenic shock, abnormal electrocardiogram, and elevated cardiac troponin I. The vessel involvement was left coronary main trunk 83.33%, left anterior descending artery 33.33% and left circumflex branch 16.67%, right coronary artery 66.67%, subclavian artery 83.33%, and renal artery 50%. Five patients received the emergency PCI. One patient died of heart failure. During follow-up 3 patients received again PCI treatment.
Conclusion: Clinical and vascular lesion characteristics of those patients were no discomfort before admission, and the suddenly typical manifestation of AMI. Severe stenosis or occlusion occurred in main coronary artery ostia and peripheral large artery. For the TA patients with hemodynamic instability the effectiveness of emergency PCI is positive.

1. Introduction

Takayasu arteritis (TA) is a rare form of chronic granulomatous large vessel vasculitis affecting the aorta and its main branches, ascending aorta, descending aorta, abdominal aorta，and pulmonary arteries. The inflammatory process results in stenosis, occlusion, dilation，or aneurysm formation in the arterial wall [1–3]. The etiology of TA is thus far undefined [4]. The Annual incidence rate ranges from 0.4 to 3.4 per million individuals and the prevalence differs from region to region [5,6]. TA is more common in Asia than that in other parts of the world. The prevalence rate of TA is from 9.0 per million to 108.3 per million individuals (9.0 per million in USA, 22.0 per million in northern Europeans, 40.0 per million in Japan, 78.1 per million in Asian whites and 108.3 per million in Africans) [2,5,7–9]. TA often occurs in young women with the age from 20 to 40 years old. The proportion of male and female patients

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is about 1:4–1:8, with 80%–90% of female patients [10–12]. Tomelleri A et al. [11] analyzed 578 patients with TA, 470 of whom were women, accounting for 81.31%. Dammacco F et al. reported 35 (81.40%) of 43 cases with TA was female [12]. Between April 2001 and March 2011, among 1372 newly registered patients with TA in Japan 83.81% (1150/1372) were female [13]. Compare female with male, female patients present as more frequently involvement of left subclavian artery, carotid arteries and aorta (ascending aorta, aorta arch, descending aorta), but male patients have higher affected rate of iliac arteries, abdominal aorta and renal arteries [11].

The early stage of TA is mainly manifested as non-specific symptoms, such as fever, night sweats, weight loss，and so on. When the lesion progresses and the inflammatory vasculitis worsens, the segmental stenosis, occlusion, dilation, and/or aneurysm formation in the arteries involvement may occur, which may result in intermittent claudication, vascular murmur, pulseless or weak pulse and decreased blood pressure，and so on [1–3]. However, it is extremely rare that TA with coronary artery involvement firstly manifested as acute myocardial infarction (AMI) at onset [14]. Huo J et al. [4] reported 5.9% (94 cases) of 1580 patients with TA (collected from January 2002 to December 2021 in several hospitals) coexisted with myocardial ischemia and neurological symptoms. Yuan SM et al. reported of 141 TA patients with coronary artery involvements (including in published 59 reports in the past two decades), 12.06% (17 cases) was diagnosed as AMI [15]. TA manifested firstly as AMI at onset is only 11 cases from January 2002 to January 2022 online retrieval with PubMed.The etiology of the coronary artery involvement is still unknown, but the pathological mechanism involves a T cell-mediated autoimmune reaction against arterial wall [16]. The clinical and vascular lesion characteristics of six patients with TA manifested firstly as AMI at onset were discussed as follows.

2. Materials and methods

2.1. Object of study

Six patients with TA manifested firstly as AMI at onset was admitted to our hospital from January 2001 to December 2019. The age of patients was 16–25 years old. Of six patients with TA, five (83.33%) were female. The patients were numbered in the order of admission. The first patient admitted was coded as number 1, and so on. Clinical findings, history, laboratory examination, angiography of the cardiovascular system, treatment, and clinical outcomes were recorded.

2.2. Diagnostic criteria of TA

The diagnosis of six patients with TA met the diagnostic criteria of American College of Rheumatology 1990 [17]：① Age of onset is less than or equal to 40 years. ② Intermittent movement disorder of limbs occurs.③ The brachial artery pulse or radial artery pulse on one side or both sides are decreased or lost. ④ The difference in systolic blood pressure between arms is greater than 10 mm Hg. ⑤ The murmurs were detected in one or both subclavian or carotid arteries.⑥ Angiography showed the segmental stenosis or occlusion of the aorta and its primary branches. The presence of 3 or more of these 6 criteria demonstrated a sensitivity of 90.5% and a specificity of 97.8%.

2.3. Indicator for observation

Medical history, primary presentation on admission, physical examination (blood pressure and heart rate), electrocardiogram, erythrocyte sedimentation rate (ESR)，levels of C-reactive protein and serum cardiac troponin I, angiographic results of the cardiovascular system, therapeutic measures, and outcomes of follow-up were recorded.

2.4. Methods and endpoints of follow-up

Angiography were used for a follow-up. The endpoints of follow-up were all-cause death, target lesion or target vessels revascularization, or recurrent heart failure (NYHA class III-IV). Follow-up ended in December 2019.

2.5. Statistical analysis

The continuous variables were expressed as mean ± standard deviation. The counting data were expressed as a percentage (%).

3. Results

3.1. Baseline data and clinical manifestations

83.33% (five cases) of six patients was female. The average age of the six patients was 20.67 ± 3.27 years old (range 16–25 years). Patient 1#, 3#, and 5# had a history of hypertension (blood pressure 150-170/110-95 mmHg) which was untreated without obvious symptoms. The other patients had no history of hypertension. None of the patients had a history of diabetes, tuberculous disease, rheumatic disease, or rheumatoid disease. No patient had the symptoms of fever, night sweats, chest discomfort, precardiac pain, arthralgia or intermittent claudication, and so on before admission, except patient 3# had intermittent effort chest tightness within 12 months before admission. All six patients presented with precardiac pain, dyspnea, hypotension, cardiogenic shock, abnormal electrocardiogram, elevated cardiac troponin I，erythrocyte sedimentation rate (ESR) increased and elevated C-reactive protein (CRP)

levels at onset and on admission (See Table 1). Patients 4# and 5# presented with ST-segment elevation myocardial infarction (STEMI). The other patients presented with extensive ST-segment depression and T wave inversion in the anterior wall leads and ST-segment elevation in the aVR lead (which was greater than or equal to 0.2 mV).

3.2. The findings of angiography

The angiography showed as follows: Patient 1# suffered from stenosis 95% in the ostium of the left main coronary trunk (LM) and stenosis 90% in the ostium of the left renal artery. Patient 2# had severe stenosis in three vessels of coronary arteries and coronary artery aneurysm (CAA) formation. Her coronary artery lesions were with stenosis 70% in LM distal, 90% stenosis in the ostium of the left anterior descending artery (LAD) and CAA after the stenosis, 90% stenosis in the ostium of the left circumflex branch (LCX), and CAA after stenosis segment, CAA in the ostium of right coronary artery (RCA) and 90% stenosis in RCA. Her bilateral proximal subclavian arteries were with occlusion 100%, abdominal aorta below renal artery was with stenosis 70%, and the bilateral proximal iliac artery was with stenosis 70–90% (see the introduction of death case). Patient 3# suffered from stenosis 95% in the ostium of LM, stenosis 90% in the ostium of LAD, 100% occlusion in the left subclavian artery (in 5 mm after opening), and 100% occlusion in the ostium of the left renal artery. Patient 4# had 90% stenosis in ostium of LM and 95% stenosis in the ostium of RCA, 100% occlusion in proximal left subclavian artery. Patient 5# suffered from 95% stenosis in the ostium of RCA, 90% stenosis in the proximal left subclavian artery, and 90% stenosis in the left renal artery. Patient 6# presented as 95% stenosis in the ostium of LM, 90% stenosis in the ostium of RCA, 90% stenosis in the proximal left subclavian artery, and 80% stenosis in the proximal internal carotid artery (see Table 2). In this group, the patients with subclavian artery stenosis and occlusions had not the obvious manifestation of subclavian artery steal syndrome.

As shown in Table 2, artery vessel involvement was LM 83.33% (5/6), RCA 66.67% (4/6), subclavian artery 83.33% (5/6), and left subclavian artery 66.67% (4/6), renal artery 50% (3/6), and 16.67% (1/6) in LCX, abdominal aorta, iliac artery and carotid artery.

3.3. Results of transthoracic echocardiography (ECHO)

Six TA patients with AMI presented with hypotension and shock on admission, so their ECHO examination was not performed before emergency PCI. At the same time of rescuing shock (using vasoactive drugs to improve organ blood flow perfusion), their emergency coronary angiography and large vessels angiography were performed. After confirming the diagnosis of TA according to the results of angiography, the emergency PCI was immediately performed to stabilize hemodynamic indicators. Except 2 # patient received ECHO of his bedside on the second day after admission, other five patients received color Doppler ECHO on the sixth to eighth day under stable condition after admission. Results of transthoracic ECHO were shown in Table 3.

3.4. Clinical treatment and follow-up outcomes

All patients presented with hypotension or shock on admission, so emergency coronary angiography (CAG) and large artery angiography were performed. Five patients (1#, 3#, 4#, 5#, and 6#) received emergency revascularization of coronary arteries and coronary stent (drug-eluting stent) implantation after the informed consent was signed by the patients and their families. Patients with subclavian artery stenosis and/or renal artery stenosis were admitted again to receive stent implantation within 2–4 months after being discharge. Five patients who received stent implantation of coronary arteries and peripheral large vessel were followed by angiography for 11–120 months (mean 26.80 ± 14.36 months). PCI again was performed due to restenosis in-stent in 3 patients (3# and 6#) with LM ostium stenosis and (5#) with RCA ostium stenosis. The rate of revascularization was 60.00% (3/5). One patient (2#) died of heart failure on the third day after admission due to refusal to PCI (see Table 4). Angiographic follow-up showed no restenosis in-stent

Table 1
Clinical features of six patients with TA manifested firstly as AMI at the onset.

Pt. No.	Sex	Age (yrs)	Manifestations before admission	Symptoms on admission	BP on Admission (mmHg)	cTn- (ug/L)	ECG	Diagnosis
1#	M	19	asymptomatic	Chest pain and dyspnea	100/50	8.1	abnormal	Extensive AMI
2#	F	19	asymptomatic	Chest pain and dyspnea	0/0	7.2	abnormal	Extensive AMI
3#	F	23	asymptomatic	Chest pain and dyspnea	100/60	6.4	abnormal	Extensive AMI
4#	F	22	asymptomatic	Chest pain and dyspnea	98/60	11.0	abnormal	Extensive AMI
5#	F	16	asymptomatic	Chest pain and dyspnea	90/56	8.6	abnormal	Extensive AMI
6#	F	25	asymptomatic	Chest pain and dyspnea	92/58	9.8	abnormal	Extensive AMI

Note: AMI = acute myocardial infarction, BP = blood pressure, cTn-I = cardiac troponin I (normal range 0 – 0.01 μg/L), ECG = electrocardiogram, ECG abnormal = ECG showed abnormal Q wave and ischemic ST-segment and T wave changes in the electrocardiogram. M = male, F = female, Pt.No = patients number, TA = Takayasu arteritis.

Table 2
The findings of angiography.

Pt. No.	LM	LAD	LCX	RCA	subclavian artery	abdominal aorta	renal artery	iliac artery	carotid artery
1#	95%	–	–	–	–	–	L90%	–	–
2#	70%	90%	90%	90%	R + L100%	70%	–	90%	–
3#	95%	90%	–	–	L100%	–	L100%	–	–
4#	90%	–	–	95%	L90%	–	–	–	–
5#	–	–	–	95%	L90%	–	L90%	–	–
6#	95%	–	–	90%	L90%	–	–	–	80%

Note: "-" = negative or not available, LAD = left anterior descending artery, LCX = the left circumflex branch, LM = left main coronary trunk. Percentage (%) in the table is the percentage of blood vessel diameter stenosis. Pt.No = patients number; Except patient 2# was with a stenotic lesion in the LM distal, all patients had stenosis in the ostium of the coronary artery. Patient 2# suffered from the occlusion in the bilateral proximal subclavian artery. R = right, L = left, RCA = right coronary artery.

Table 3
Results of transthoracic echocardiography (mm).

Pt. No.	LA	AO	RVOT	RV	IVS	LV	LVPW	EF (%)
1#	25	25	22	18	11	45	11	62
2#	30	28	24	16	11	52	9	40
3#	26	26	22	18	11	43	11	68
4#	25	25	23	18	9	44	9	69
5#	28	28	26	18	11	46	11	68
6#	28	28	26	18	11	45	11	67

Note: AO = aortic root diameter; EF (%) = left ventricular ejection fraction (%) , IVS = interventricular septal thickness, LA = left atrium diameters, LV = left ventricle end-diastolic diameters, LVPW = left ventricular posterior wall thickness, RV = right ventricle; RVOT = right ventricular outflow tract.

in patients with peripheral large vessel stent implantation.

3.5. Introduction of death case

Patient 2# was a maiden girl (19 years old) and suffered from large vessels lesions of multiple organs. She had always felt herself in good health. There was no history of joint pain, low fever, night sweats, etc. She said she often went swimming and sometimes felt weak in her upper limbs. She had no previous hypertension, tuberculosis, hepatitis, joint disease, diabetes, rheumatic and rheumatoid diseases, etc. Because of a quarrel with her family, she suddenly felt chest pain and dyspnea, which lasted for 2 h and did not relieve, so she had to admit into our hospital. The patient's height was 160 cm, weight 53 Kg, body temperature 36.8 °C. His radial artery pulse of both upper limbs was not palpable. The pulse of the bilateral dorsalis pedis artery was not palpable. The blood pressure of both upper limbs was 0/0 mmHg. Her body was developed normally, well-nourished, conscious, and expressionless and distressed face. His jugular vein was no dilatation. The heart rate was 80 beats/min. The first heart sound was decreased. There was no obvious murmur in each valve area. The breath sounds of both lungs were clear and without rales. His abdomen examination and neurological tests showed no abnormalities. ECG shown (see the supplementary figure 2) that ST-segment elevation in I and aVL leads was 0.15–0.20 mV, ST-segment depression in II, III and aVF leads were 0.20–0.30 mV, and R wave voltage of V1–V3 was significantly decreased (0.05–0.1 mV). Transthoracic echocardiography showed left atrium (LA) 28 mm, aorta (AO) 28 mm, right ventricular outflow tract (RVOT) 20 mm, right ventricle (RV) 14 mm, interventricular septum (IVS) 11 mm, left ventricle (LV) 52 mm, left ventricular posterior wall thickness (LVPW) 9.0 mm and left ventricular ejection fraction (EF) 40%. Laboratory examination showed no abnormality in routine hematuria, serum ions, thyroid function, liver function , renal function, and blood lipid examination. Other tests were shown in Table 5.

Table 4
Clinical treatment of large vessel involvements and follow-up outcomes.

Pt. No.	coronary artery	subclavian artery	abdominal aorta	renal artery	iliac artery	carotid artery	FU period	FU outcomes
1#	stenting	–	–	Stenting	–	–	45	normal
2#	not treated	not treated	not treated	–	not treated	–	3D*	death
3#	stenting	stenting	–	stenting	–	–	37	again P
4#	stenting	stenting	–	–	–	–	26	normal
5#	stenting	stenting	–	stenting	–	–	15	again P
6#	stenting	stenting	–	–	–	no	11	again P

Note: "-" = negative or not available, 3D* = the patient died of heart failure on the third day after admission. again P = because of restenosis in-stent the patient once again received PCI. FU = follow-up. not treated = the patient did not receive PCI treatment, PCI = percutaneous coronary intervention, Pt.No = patients number, Stenting = the patient underwent PCI and stent implantation.

Angiographic findings of coronary arteries and large artery vessels (see Figs. 1–6, and see the supplementary video picture 1).

The patient was diagnosed with Takayasu's Arteritis (Type III [18]) , non-ST-elevation AMI, LM and three coronary artery stenotic lesions and coronary artery aneurysm, bilateral subclavian artery occlusion, abdominal aorta stenosis with thrombosis, right external iliac artery stenosis, left heart dysfunction, cardiogenic shock and cardiac function NYHA class IV. The patient received the treatment of the antishock (fluids and vasoactive drugs were administered), antithrombosis (aspirin, clopidogrel and heparin sodium) , anti-inflammatory (penicillin) and immunosuppressant (cyclophosphamide) and glucocorticoids (dexamethasone), but rejected PCI and died of aggravated heart failure on the third day after admission.

4. Discussion

4.1. Diagnosis about TA

American College of Rheumatology 1990 criteria for the classification of Takayasu arteritis [4] was used to diagnose TA. This criterion has good sensitivity and specificity. However, large vessel angiography has become the gold standard for the diagnosis of TA. Computer tomography angiography (CTA), magnetic resonance angiography (MRA), ultrasonography and positron emission tomography could not only provide important information for early diagnosis but also detect disease activity, and thus further guide the treatment in TA. The diagnosis of TA can be made when angiography reveals localized or segmental stenosis or occlusion of the aorta and its major branches and/or pulmonary artery, or aneurysm formation and thickening of the vessel wall as indicated by intravascular ultrasound based on the characteristic of age at onset. All 6 patients in the study received coronary angiography and large vessel angiography and were confirmed to suffer from TA. This result suggests that it is should be considered that the etiology of young AMI patients may be large vessels arteritis or TA, and large vessel angiography further should be performed to avoid missing the important cause and diagnosis of large vessels arteritis. The incidence of coronary arteries involvements in TA was reported to be about 9–10% [1]. AMI as the first manifestation of TA is rare. Although young women rarely suffer from AMI, TA is one of the main causes [19]. Spontaneous coronary artery dissection (SCAD) also is a significant cause of myocardial infarction in women and account for less than 1% of AMI cases. SCAD occur most often between the ages of 47 and 53 years [20–22]. 90% of SCAD occur in perimenopausal, pregnancy and postpartum women with fibromuscular dysplasia [22]. CAG can clearly diagnose SCAD and shows multiple false vascular lumens or intramural hematoma in LAD, LCX or RCA. In the autopsy of a patient who died of SCAD [23] pathological examination showed eosinophil infiltration in epicardial coronary artery wall, which was limited to adventitia and periaortic soft tissue. These characteristics are helpful to differential diagnosis with TA.

4.2. Clinical features of TA with AMI as the initial presentation

Patients with AMI as the initial presentation of TA is rare, and only sporadic case reports. Cavalli G et al. reported among 1950 female patients aged <40 with chest pain, dyspnea, palpitations, angina, heart failure, or cardiac arrest, 40 suffered from acute ischemic heart disease. The classic' atherosclerosis was 60% (24 cases) and TA was only 10% (4 cases) [6]。In this study, the six TA patients (who manifested firstly as AMI at onset) had four main clinical features. (1) 83.33% (5/6) of the patients had no symptoms before onset. Only one patient had effort angina within one year before the admission, which could be relieved by getting some rest or

Table 5
Laboratory findings.

Items	Test values	Normal range
ESR	58.00 mm/h	0–20 mm/h*
ASO	450.00 u/L	0-25 u/L
RF	19.00 u/L	0-15 u/L
CRP	18.00 mg/L	0–10 mg/L
LDH	684.00 u/L	135-240 u/L
AST	352.00 u/L	0-40 u/L
CK	2934.00 u/L	26-174 u/L
CK-MB	200.00 u/L	0-25 u/L
a-HBDH)	323.20 u/L	72-182 u/L
ALT	52.00 u/L	0–40 U/L
Uric	253.00 μmol/L	150-350 μmol/L
cTn-I	50.00 ng/ml	0–0.01 ng/ml
BNP	244.00 pg/ml	0–100 pg/ml
ANA	Negative	Negative
HIV	Negative	Negative
CCP	Negative	Negative

Note: ALT = alanine aminotransferase, ANA = antinuclear antibody, ASO = antistreptolysin O, AST = aspartate aminotransferase, BNP = brain natriuretic peptide, CCP = anti-cyclic citrulline peptide antibody, CK = phosphocreatine kinase, CK-MB = creatine phosphokinase-isoenzyme-MB, CRP = C-reactive protein, cTn-I = cardiac troponin I, ESR = erythrocyte sedimentation rate, a-HBDH = a-hydroxybutyrate dehydrogenase, HIV = human immunodeficiency virus, LDH = lactate dehydrogenase, RF = rheumatoid factor, Uric = uric acid.

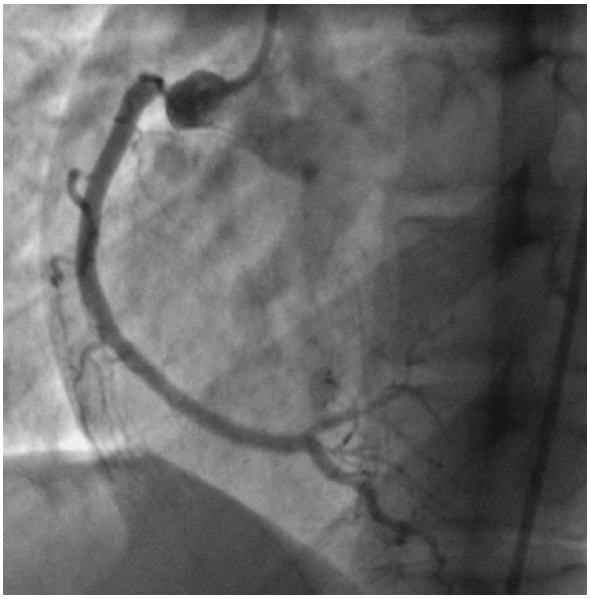

Fig. 1. RCA proximal aneurysm in RCA ostium, and stenosis 95% in RCA.

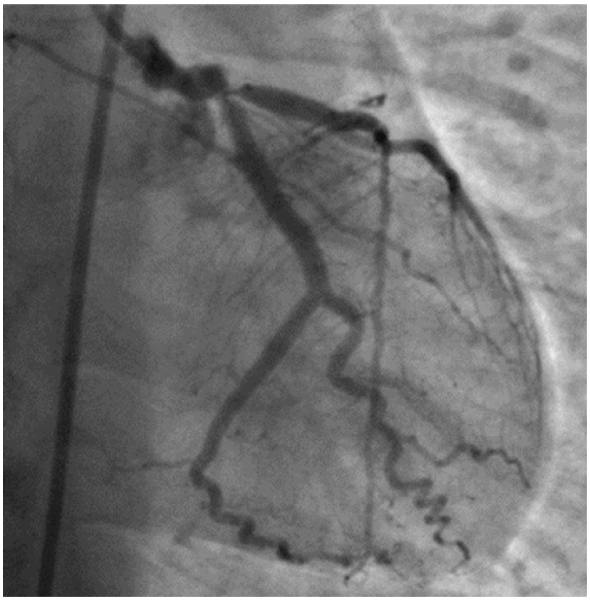

Fig. 2. Stenosis in distal LM and in ostia of LAD and LCX.

taking nitroglycerin (sublingual administration), and was without other symptoms. (2) Female patients accounted for 83.33% (5/6). (3) 100% of patients manifested suddenly as chest tightness and pain, dyspnea, hypotension, or cardiogenic shock. (4) 66.67% (4/6) patients presented with non-ST-segment elevation AMI and 83.33% (5/6) patients presented with the extensive anterior wall AMI.

4.3. Features and pathogenesis of coronary artery involvements

The proportion of coronary arteries involvement in patients with TA is about 9–10% [1,4,5,14,24]. TA mainly involves the ostia and proximal segment of coronary artery, showing the lesions of stenosis, occlusion or dilatation, which result in myocardial ischemia, angina pectoris or AMI [4,25]. Six cases described in the study all suffered from the ostia involvement of the LM and/or RCA. Features of coronary artery lesions showed that (1) the coronary arteries lesions mainly represented as stenotic lesions or aneurysms in ostia of coronary artery originating from aorta root. In this study patient 1# was with 95% stenosis in LM ostium, 2# with severe stenosis and

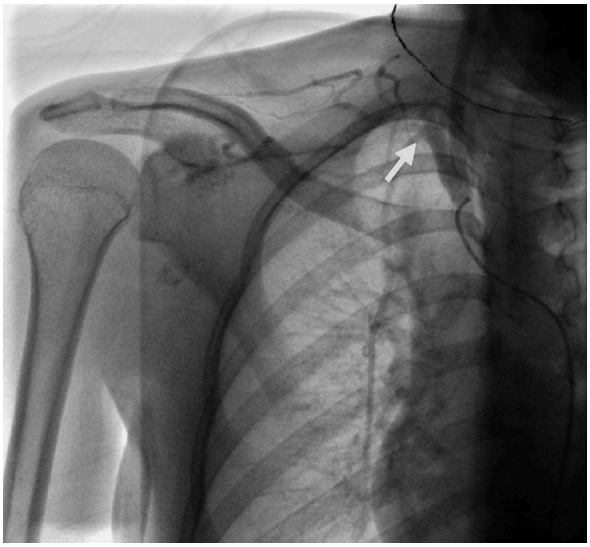

Fig. 3. Occlusion of right subclavian artery (arrow).

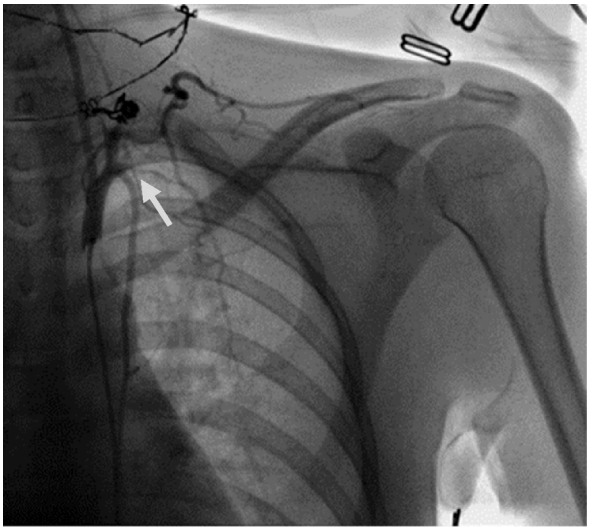

Fig. 4. Occlusion of left subclavian artery (arrow).

aneurysm in ostia of LAD, LCX, and RCA, 3# with 95% stenosis in LM ostium, 4# with 90% stenosis in LM ostium and with 95% stenosis in RCA ostium, 5# with 95% stenosis in RCA ostium, and 6# with 95% stenosis in LM ostium and 90% stenosis in RCA ostium. The occurrence of such coronary artery lesions is caused by the aortic root inflammatory changes associated with aortitis, and is an inflammatory manifestation of the ascending aorta [14]. The autopsy showed that inflammation caused a significant thickening of the aortic and coronary artery walls and abundant granulomatous and a lymphoplasmacytic infiltrate in the vessel wall [26]. The avascular necrosis of the vascular wall may also occur [27]. (2) LM is the most common in coronary artery involvement. In this study, 83.33% (5/6 cases) was the lesions in LM ostia. Among 5 patients with LM lesions, 4 (80.00%) had LM ostia stenosis, and only 1 patient (No. 2) had LM terminal stenosis. All patients with LM lesions presented with extensive AMI and shock. Since RCA originates directly from the aorta, inflammation of the aorta often affects the RCA opening. In this group, RCA ostium was affected in 66.67% (4/6 cases), LAD ostium was involved in 33.33% (2/6 cases) and LCX ostium was involved in 16.67% (1/6 cases). Most of the coronary arteries involvements presented with localized stenosis and a few presented with coronary aneurysm formation. These characteristics are helpful to distinguish coronary stenosis caused by coronary atherosclerosis. The stenosis of coronary artery opening, under the interaction of internal and external pathogenic factors (such as fatigue , mental tension and stress, sympathetic nerve excitation, coronary artery spasm, inflammation, elevated blood sugar, and elevated blood uric acid , smoking, drinking, dyslipidemia etc. [4, 28]) will make the rapid progress of the lesion, the rapid aggravation of stenosis, resulting in acute ischemic necrosis of the myocardium, and the occurrence of AMI. The pathogenesis of coronary arteries involvement with TA is still unclear so far. Recently

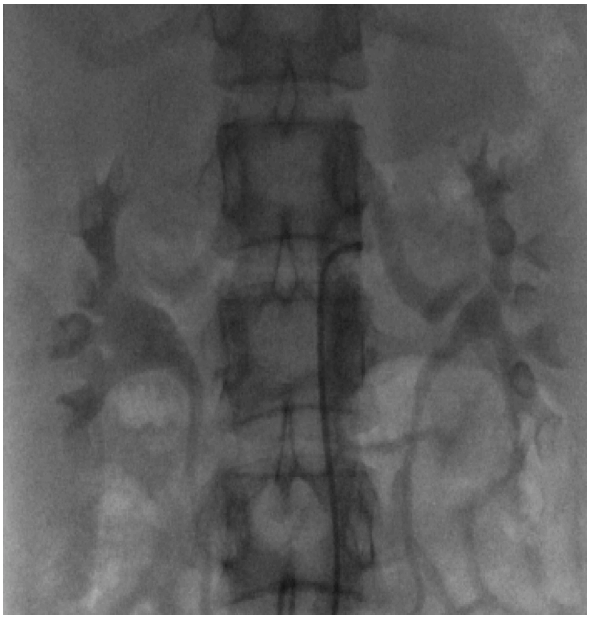

Fig. 5. There was no stenosis of the bilateral renal arteries.

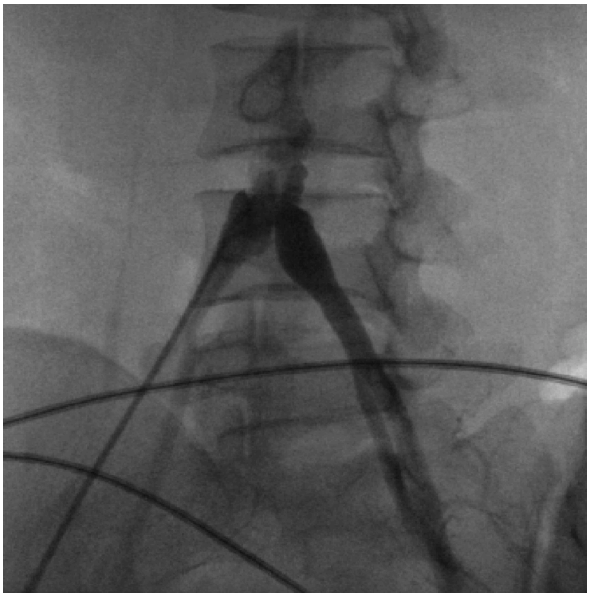

Fig. 6. Abdominal aortic stenosis was associated with thrombosis in 70%. Right external iliac artery stenosis 95%.

study reported HLA-B*52 allele was significantly associated with TA. However, the ostium involvement of the coronary arteries is caused by the extension of inflammation of the aortic root or ascending aorta [14,24,29], which leads to arterial wall remodeling, thickening, aneurysmal lesions, and stenosis or occlusion of the coronary vessel lumen [24,26,29]. In the acute inflammatory phase (active stage) of TA proinflammatory T-cell cytokines (such as TNF-α, IL-6 and IFN-γ, etc.) may be associated with granuloma formation in the tissue of aortic wall. Inflammation begins to invade the vasa vasorum in early stages of TA, which cause an inflammatory cell infiltration of adventitia [5]. Patients become increasingly infectious as their illness progresses. A lot of monocytes, lymphocytes, neutrophils, epithelioid cells, and giant cells infiltrate to tissue lesions from adventitia to and internal elastic lamina of coronary artery, which eventually results in granulomas formation and intimal hyperplasia, and cause to stenosis or occlusion of the arterial lumen and ischemic symptoms [29,30].

4.4. Characteristics of large artery vessel involvements

Characteristics of patients with AMI as the first manifestation of TA also include systemic large vessel involvements. It is an important condition or clue for the diagnosis of TA that coronary artery lesions combined with large vessel lesions (stenosis, occlusion or dilation) in other organs of the body. Rarely, TA is only involved in the coronary artery. Most of the patients with TA have coronary artery lesions combined with stenosis of proximal subclavian artery, renal artery-opening, carotid artery or aorta. Subclavian artery stenotic lesions are the most common. In this study, six patients with TA all presented as peripheral large vessel involvement. Subclavian artery stenosis occurred in 83.33%. The left renal artery was involved in 50.00%. Bilateral subclavian artery stenosis or occlusion is rare. Patient 2# in this study had bilateral subclavian artery occlusion, which was very rare.

4.5. Treatment and prognosis

Treatment for the stenosis or occlusion of coronary artery involvements in TA includes percutaneous coronary intervention (PCI) and coronary artery bypass grafting (CABG). PCI includes percutaneous transluminal coronary angioplasty (PTCA) and stent implantation. Because these TA patients who manifested firstly as acute myocardial infarction at onset would have hemodynamic instability or shock, PTCA can quickly open the occluded coronary arteries and reduce the coronary stenosis degree, improve coronary blood flow and save dying myocardium, which is conducive to relieving symptoms [31]. However, PTCA alone has a high risk of acute re-occlusion and long-term restenosis of the target vessel. The long-term restenosis rate of the target vessel after bare metal stent (BMS) implantation was also high. Drug-eluting stent (DES) implantation can inhibit intimal proliferation and progression of vascular wall fibrosis, so the restenosis rate is significantly lower than that of PTCA and BMS implantation. TA with AMI as the first manifestation at onset is an acute and severe illness with hemodynamic instability and requires emergency treatment. Therefore, emergency PCI is the main treatment to save life of this kind patients. Emergency PCI has good efficacy and high safety and is the preferred treatment for patients with hemodynamic instability. In this study, except for one patient (2#) who died of refusing PCI, the other 5 patients received emergency PCI and DES implantation to improve the disease. Patient 2# presented as severe hemodynamic disorder and cardiogenic shock on admission (see 2.4 Introduction of death case). Her echocardiography showed left ventricular enlargement and decreased ejection fraction (see Table 3). His CAG showed coronary stenotic lesions in the ostia of RCA, LAD and LCX (see Figs. 1 and 2), and stenosis of LM terminal (see Fig. 2), which leads to myocardial ischemic injure and necrosis and cardiac dysfunction. The patient and his family absolutely refused emergency PCI to lead his death. Studies have shown that CABG is superior to PCI, and is with lower rates of restenosis and complications [15,32]. However, the high mortality of emergency CABG for patients with AMI, especially those with hemodynamic instability, makes it inappropriate to be the preferred approach for these patients. Unfortunately, DES and CABG both are less effective in patients with TA than that in patients with atherosclerotic lesions. This phenomenon may be related to persistent inflammation action of the blood vessel wall in patients with TA [4,18]. PCI is the first choice for TA patients with acute myocardial ischemia or AMI. Vascular surgery remains an important option in the management of TA with stable hemodynamics [26]. Huo J et al. [4] reported that the mortality rate in the conservative treatment group was significantly higher than that in the interventional management and surgical treatment groups during a mean follow-up of 57.79 months.

Renal artery stenosis or subclavian artery stenosis caused by TA can also be treated by stenting [33]. In this group, all patients with renal artery involvement or subclavian artery stenosis or occlusion underwent selective BMS implantation. The diameter of stent implantation was 6–8 mm [34]. The incidence of restenosis in-stent was low due to the large diameter of the stent implantation. During follow-up, no restenosis in-stent occurred in 5 patients with renal artery and/or subclavian artery stenosis or occlusion. This result indicates that stent implantation in large artery vessels is effective. Pharmacologic treatment for TA includes antithrombotic therapies, glucocorticoids, and immunosuppressive agents [35,36]. Methotrexate is recommended to use for the treatment of all patients with TA [37]. In recent years, biological drugs have been widely used in clinical practice. Tumor necrosis factor α inhibitors are most commonly used, such as infliximab and adamuzumab, which can inhibit the development of active inflammatory lesions [38,39].

5. Limitations of this study

The clinical and vascular lesion characteristics of six patients with Takayasu arteritis manifested firstly as acute myocardial infarction at onset were studied. Because the cases are very rare, the sample size of this study is small. Further studies with larger sample sizes are needed to support the results.

6. Conclusion

Comprehensive the above , the patients with TA manifested firstly as AMI is very rare. The clinical and vascular lesion characteristics of those patients were that most of the patients had no discomfort before admission, and suffered from the suddenly typical manifestation of AMI and hemodynamic instability at the onset. Angiography showed severe stenosis or occlusion of LM ostia, RCA ostia, and peripheral large artery. This suggests that for young patients with AMI, CAG should be accompanied by large artery angiography to improve the diagnostic rate. For the TA patients with hemodynamic instability the effectiveness of emergency PCI is positive.

Authors' contributions

RFL conceived and supervised the study, was involved in the PCI procedure and wrote the main body of the manuscript. FXX collected the clinic data and participated in the discussion on the interpretation of the research content. YJZ directed the drafting of the manuscript and critically revised the manuscript. TKL took part in the PCI procedure, performed statistical analysis of the data, and revised this paper. All authors critically revised and approved the final version of the manuscript.

Declarations

Ethics approval and consent to participate

All participants provided written consent before entering the study, and approval was obtained from the Ethics Committee of Beijing Anzhen Hospital, Capital Medical University, Beijing.

Availability of data and materials

All data generated or analyzed during this study are included in this published article and its supplementary information files. Data other than these are available from the corresponding author on reasonable request.

Conflicts of interest

The authors declare that there is no conflict of interest regarding the publication of this article.

Statement

The authors state that all methods used in this study were carried out in accordance with following guidelines and regulations and guided by the following guidelines. That is "2018 Update of the EULAR recommendations for the management of large vessel vasculitis (Hellmich B et al. Annals of the Rheumatic Diseases. 2020; 79:19–30.), Management of Takayasu arteritis: a systematic literature review informing the 2018 update of the EULAR recommendation for the management of large vessel vasculitis (Águeda AF et al. RMD Open. 2019; 5 (2):e001020.), and 2021 American College of Rheumatology/Vasculitis Foundation Guideline for the Management of Giant Cell Arteritis and Takayasu Arteritis. (Maz M et al. Arthritis Rheumatol. 2021; 73 (8):1349–1365.), and Diagnostic criteria for Takayasu arteritis (Sharma BK et al. Int J Cardiol. 1996; 54 Suppl: S141-7)".

Abbreviations

AMI = acute myocardial infarction, BMS = bare metal stent, CAA = coronary artery aneurysm, CABG = coronary artery bypass grafting, CAG = coronary angiography, CRP = C-reactive protein, CTA = computed tomography angiography, cTn-I = cardiac troponin I, DES = drug-eluting stent, ECG = electrocardiography, ECHO = echocardiography, ESR = erythrocyte sedimentation rate, LAD = left anterior descending artery, LCX = the left circumflex branch, LM = left main coronary trunk, MRA = magnetic resonance angiography, NYHA = New York heart association class of cardiac function, PCI = percutaneous coronary intervention, PTCA = percutaneous transluminal coronary angioplasty, RCA = right coronary artery, SCAD = spontaneous coronary artery dissection, STEMI = ST-segment elevation myocardial infarction.

Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.heliyon.2023.e13099.

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